The state of lipid metabolism, lipid peroxidation and antioxidant defense in patients with chronic obstructive bronchitis with hypertrophy and atrophy of the myocardium

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Introduction. In recent years, there has been an increase in the proportion of morbidity and mortality of population from chronic non-specific lung diseases (CNSLD) [1]. This is due to a number of reasons: air pollution, smoking, resistance of the bacterial flora to some antibiotics. A special attention of public health authorities to this problem is determined by the fact that the consequences of CNSLD, complicated by pulmonary heart disease (PHD), eventually lead to patients' (primarily men) disability and death at able-bodied age (50-60 years) [2]. Thus, according to the WHO Expert Committee, more than half of patients with lung diseases develop PHD, which leads to frequent hospitalization. Statistics show that 16-20% of all hospitalized subjects are PHD patients [3].

According to modern research findings, activation of lipid peroxidation is one of the leading links in PHD pathogenesis in chronic bronchitis [4].

The aim of our research was to study the dynamics of markers of lipid peroxidation (LPO) and antioxidant defense (AOD) in patients with chronic obstructive bronchitis (COB) and heart failure (HF) with myocardial hypertrophy and atrophy.

Materials and methods. To solve the abovementioned tasks, we examined 62 patients of both sexes with COB and HF, stage I-II. Each of them had at least 2 risk factors for COB (smoking, dustiness of the workplace, frequent colds, family history). Determination of nosological form of COB was carried out in accordance with the WHO criteria. The stage of circulatory failure was assessed in accordance with the classification by N.D. Strazhesko and V.Kh. Vasilenko. The diagnosis of COB was made on the basis of anamnestic, clinical and biochemical, instrumental data obtained by examining patients.

30 patients (study group 1) with hypertrophy of the myocardium and 32 patients with myocardial atrophy (study group 2) were examined.

All patients underwent the following tests: total serum lipids, total cholesterol, β-lipoproteins, phospholipids, peroxide hemolysis of erythrocytes (PHE), malonic dialdehyde (MDA), superoxide dismutase (SOD), ceruloplasmin, catalase according to standard methods.

Results. Our findings indicate that in both groups of patients, the total lipids, cholesterol, and β-lipoproteins in the blood were significantly increased. Studies of LPO in these groups of patients showed an increase of MDA in both groups of patients, but in the group with myocardial atrophy these rates were significantly higher than in the group with hypertrophy (14.34 ± 0.28 μmol / l and 10.67 ± 0.55 μmol / l, respectively). The increase in peroxide hemolysis of erythrocytes was also more manifested in the group with myocardial atrophy (19.38 ± 1.05%) than in the group with hypertrophy (14.11 ± 0.89%). A statistically significant ($p<0.05$) decrease

in catalase activity in the group with myocardial atrophy (2.11 ± 0.07) was also revealed. From the data obtained it follows that a greater activation of LPO was noted in the group with myocardial atrophy. This can be associated with the release of pro-oxidant factors from the destroyed cardiomyocytes.

Conclusion. Thus, the change in LPO markers indicates its significant activation in the development of myocardial atrophy, which makes it possible to consider the activation of LPO as one of the leading mechanisms in the development of this particular form of PHD in patients with CNSLD.

Prospects for further research. The obtained results are the basis for the development and application of schemes for the diagnosis and treatment of PHD in patients with CNSLD, focused on antioxidant therapy.

Recommendations. It is advisable to use LPO markers to evaluate the course of CNSLD with PHD.

Key words: chronic non-specific lung diseases, pulmonary heart disease, heart failure, hypertrophy of the myocardium, myocardial atrophy, lipid peroxidation, antioxidant defense.

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